OSTEOMALACIA—REPORT OF A CASE.

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Osteomalacia is a metabolic disturbance, characterized chiefly by decalcification of the bones. As a result of the softening of the affected bones, particularly those of the spinal column and pelvis, distortion and compression take place, with secondary nerve root irritation. Referred pain, therefore, is a prominent symptom of osteomalacia.

The following case is reported as a typical example of the disease. Attention is called to the fact that the first symptom was pain; and pain continued to be the predominant symptom until recalcification of the bones was well under way.

The patient was a white female, age 48, married five years before the onset of the present illness; never pregnant.

Present Illness.

The onset was insidious and began while the patient was in Florida. The first symptom was pain in the left thigh and knee, especially noticeable when ascending steps. This symptom developed in February of 1929. During the following six weeks the pain increased in intensity and extent, eventually involving the neck and shoulders, and radiating through the lower abdomen and down both legs. In April, about two months after the onset of the pain, an abdominal operation was performed and a "fecal appendix" removed. While in bed, after the operation, the pain subsided, but after being up one week, it recurred, and then for the first time, she developed vague intestinal disturbances, characterized chiefly by sensations of gas in the lower bowel, bloating, loss of appetite, vague nausea and occasional vomiting. There was no diarrhea or change in the appearance of the stools. At this stage pellagra was suspected. During the next two months the patient became rapidly and progressively worse. The chief complaint was of pain, which became generalized from the head to the feet, and was intensified by the slightest motion, rendering her completely helpless. The loss of appetite was extreme, and

the bloating and nausea were very annoying. She also developed a waxy pallor and a peculiar "staring" expression.

On August 1, six months after the first symptom, the patient's sister arrived from the North and devoted her time and energy to encouraging the patient to eat, particularly all forms of green vegetables. Under this green vegetable diet, improvement was definite, so that in three weeks it was possible for the patient to sit up and even take a few steps. Motion, however, caused considerable pain, especially movements of the neck, back and hips. At this time, when the patient was gotten up, it was first noticed that there was marked shortening of the neck.

The patient was brought to her home in the North and placed in a sanitarium. She continued to improve for several months, then relapsed and became practically helpless because of the pain on motion. The gastrointestinal symptoms likewise returned. The loss of weight from the time of the onset of the illness was 70 pounds. Whether this loss had been gradual or had occurred intermittently was not known.

Past History.

The past history was essentially negative. The patient had always been unusually well. Previous to her illness, her high color, erect bearing, and physical vitality had been such as to draw comment. There was no acute or chronic illness, nor was there any history of goitre or metabolic disturbances. The menstruation was regular, accompanied by moderate pain; but since the onset of the present illness, menstruation was painless, irregular, intervals varying from three weeks to three months, flow varying from scant to profuse. Whether this menstrual irregularity was due to the onset of the menopause or the effects of the illness was not clear.

Physical Examination.

When seen on March 1, 1930, the patient was pale, waxy, and there was a slight generalized subcutaneous edema, but no pigmentation. She could tolerate slow, passive movements and could be gotten out of bed and propped up in a chair without precipitating any severe pain. It was evident, however, that the patient held herself rigidly, permitting almost no movement of the head, neck, back or hips. Any attempt to exceed, by passive motion, the very slight

limits, caused excruciating pains. The neck was shortened and the head forward. There was a moderate exaggeration of the normal dorsal curvature, and a flattening of the lumbar curve. The reflexes were all normal and aside from a moderate hyperesthesia, there were no sensory disturbances. There was no muscle atrophy. The eyes, ears, throat, heart and lungs were all normal. Examination of the abdomen revealed general tympany and hyperperistalsis, but no other abnormality. The blood pressure was 120/80.

On March 17, 1930, the patient was admitted to Magee Hospital for study. X-rays showed numerous small areas of decalcification involving the bones of the entire spine, the bones of the pelvis, both shoulder and hip joints, and the ribs. There was no evident involvement of the bones of the skull or the shafts of the long bones. There was considerable flattening and distortion of the cervical vertebrae and to a less extent of the dorsal and lumbar vertebrae.

On the basis of the clinical signs and X-ray evidence, a tentative diagnosis of osteomalacia was made. The subsequent course of the patient confirmed this diagnosis.

LABORATORY STUDIES.

March 18, 1930: R.B.C., 3,100,000; H.B., 60%; W.B.C., 5,700.

Differential: P.M.N., 47; large monos, 40; small monos, 4; transitionals, 5; basophils, 1; myelocytes, 3; nucleated reds, occasional.

March 18, 1930: Blood N.P.N., 31.4 mg. per 100 c.c.; blood sugar, 74 mg. per 100 c.c.

March 22, 1930: Wassermann and Kahn test negative.

March 24, 1930: Bence-Jones protein in urine—no trace.

March 30, 1930: Blood calcium, 10 mg. per 100 c.c.

Urine: Routine examination negative at all times. Repeated tests for Bence-Jones protein were negative.

Pelvic Examination: (Doctor Chalfant) Pelvic organs normal.

Temperature: Normal throughout.

Pulse: 100-110 during the first week. April 1-7th, 90-100; April 7 to June 30th, 80-90.

Treatment: Three lines of treatment were decided upon; first, sterilization by deep X-ray over the ovaries; second, high vitamin diet, especially vitamin D, together with calcium and phosphorus;

third, orthopedic measures designed gradually to correct the deformities of the spine.

The X-ray sterilization was promptly effective. No secondary nervous phenomena developed.

The vitamin D was administered as Viosterol, 10 drops twice daily, and ultraviolet radiation was given daily. The phosphorus was given in the form of Phytin (Ciba), three to six tablets daily. In about six weeks an intolerance to this developed, as shown by a tendency to vomit, and thereafter it was given only intermittently.

The calcium was given by mouth, in various forms. Two injections of 10 c.c. of 10% calcium gluconate were given intravenously (5/18 and 5/24).

An important element in the treatment was reassurance and encouragement. The nature of her condition was explained to the patient, and with the outlook of a cure she lost much of her depression and nervousness, and willingly coöperated in attempting to eat a high vitamin diet. Many of her gastric symptoms promptly subsided, due in large part, we thought, to her improved morale.

The orthopedic measures consisted in moderate traction, collar support, etc., while in bed; and after the patient was able to be up, a brace to support the entire spinal column.

Clinical Course.

Under the above regime the patient improved steadily. The pain diminished and was entirely gone by the end of eight weeks. The appetite improved, the anemia disappeared, and during August the patient was gotten out of bed for increasing intervals.

X-rays taken on May 20th, 1930, showed no evident recalcification, although the patient was almost free from pain. On June 24th, 1930, however, X-ray of the cervical and pelvic bones showed considerable recalcification; in some places the bones being even more dense than normal.

The patient left the hospital on September 6, 1930, markedly improved. She was free from pain, able to walk short distances with the aid of crutches; her color was normal, blood count normal, and all nervousness had disappeared.

The residual deformity was not great. Her stature was decreased 5 inches (from 65 to 60) and there was evident shortening of the neck

with considerable limitation of movement of the neck. There was a moderate exaggeration of the normal dorsal curve and some flattening of the lumbar curve, but no gross change of the pelvis. The arms and legs were straight.

Since discharge from the hospital, improvement has continued. Recent X-rays showed marked calcification of the affected bones. The patient has discarded the orthopedic braces and walks without difficulty. In a recent communication she states that she is now driving her car. She has regained 40 pounds in weight.

Discussion.

Osteomalacia is a bone affection that is rare in the United States. It is much more common in Switzerland and northern India, and it may be of some significance that it seems to occur in certain districts, as is the case with endemic goitre. It is a disease that occurs usually in early adult life, and is almost limited to females, various reported series showing an incidence of 2 per cent to 10 per cent males.

Pathologically the disease is characterized by a widespread softening and absorption of pre-existing bone and the formation of uncalcified, new osteoid tissue. By reason of this softening, marked deformities occur through bending, flattening or fracturing.

The predominant symptoms are pain and muscular weakness. The affected bones are tender, but much of the pain in the case here reported seemed to be due to the nerve root irritation incident to the flattening and distortion of the vertebrae.

The etiology of osteomalacia is unknown. The high incidence of females, and the fact that in many cases there is a history of repeated pregnancies, has led many to the conclusion that the glands of internal secretion, especially the ovaries, are at fault. McCrudden,[1] however, after an exhaustive study, concludes that the ovaries are not at fault.

Of particular interest in connection with osteomalacia, are the recent reports on the bone changes accompanying overactivity of the parathyroid glands.[2] These cases reported show decalcification, which results in bony changes similar to those found in osteomalacia, osteitis fibrosa, and, in one instance[3] a concurrent benign giant cell sarcoma.

During the active stages of these cases of hyperparathyroidism,

there is a high calcium content of the blood (14 mg. to 20 mg. per 100 c.c.); increased urinary output of calcium, and a negative calcium balance; diminished blood phosphorus; atonic condition of the muscles; and, in some cases, deposition of calcium in abnormal places, as in the form of a kidney stone.

In the majority of the cases reported, the removal of parathyroid tumors was followed by remarkable cure, the blood chemistry returning to normal and the bones promptly recalcifying.

In conjunction with these clinical observations the experimental work reported by Jaffe[1] shows that the bone changes typical of osteitis fibrosa can be produced in guinea pigs or dogs by injecting large amounts of parathormone.

Since Collip isolated and studied the effects of the hormone of the parathyroid gland, it has been well recognized that parathormone mobilizes the calcium of the body. The recent studies, cited above, would indicate that this increased calcium content of the blood is obtained from the bones, and that the eventual effect on the bones is a picture that varies somewhat, in some cases being typical of osteomalacia, in other cases, characteristic of osteitis fibrosa, or of Paget's disease, or even of giant cell sarcoma.

Perhaps of significance also in relation to osteomalacia is the report of E. V. McCollum, before the recent Meeting of the American Society of Biological Chemists, at Montreal, on the effects of an insufficient intake of magnesium. His experiments show that a complete absence of magnesium from the diet of the rat resulted in such a drainage of calcium and phosphorus from the body that not enough was left to permit an X-ray visualization of the skeleton. Other signs that occurred led McCollum to conclude that there is a relation between magnesium and the adrenal glands.

It is evident, therefore, from the recently acquired knowledge of the effects of the parathyroid secretion, that hyperparathyroidism can cause osteomalacia. However, not all cases of this disease can be attributed to the parathyroids. The case here reported was not accompanied by any increase in blood calcium; and her recovery would lead one to conclude that the parathyroids were not abnormal.

A consideration of the cases of osteomalacia reported indicates that the condition may arise from more than one cause. Any disturbance that depletes the body of calcium may result in the bony

changes typical of this disease. Thus, one must consider among etiological factors a prolonged insufficient calcium (or magnesium) intake; an abnormal demand for calcium (as in pregnancy and lactation); and, finally, the various endocrine disturbances, particularly of the parathyroids.

Clinically, the predominant symptom of osteomalacia is pain. In the early stages, before the bone changes are evident, the severe pain may be entirely unexplainable. Moreover the tendency of the process to localize in certain areas may add to the difficulties of diagnosis. Thus, the earlier pains may be limited to one extremity or to the pelvis, or abdomen. It may be that milder cases may show local pain, which later subsides with spontaneous recovery, and the true condition thus never be recognized. The persistence of unexplainable pain, especially if it is of the referred type, should lead to an examination of the bones for tenderness or X-ray evidence of decalcification.

The treatment depends somewhat on the available evidence as to an etiological factor. Should there be evidence of a hyperparathyroidism, parathyroidectomy is indicated. In the presence of normal blood calcium, the treatment consists of a diet rich in vitamins A and D, ultraviolet radiation, and calcium and phosphorus. Whether obliteration of the ovaries (by X-ray) was in any way responsible for the improvement of the patient here reported, cannot be judged. Her recovery may have been the result of the other measures instituted.

The orthopedic aspects of osteomalacia are difficult and require constant attention. The prevention or correction of deformities, resulting from the softened bones requires ingenuity and skill. Moreover, orthopedic measures may relieve local pressure on nerves, and thus markedly ameliorate the intense suffering.

Conclusion.

A case of osteomalacia is reported in which the typical bone changes caused severe pain and moderate deformity.

Treatment consisted of: (a) sterilization by exposure of the ovaries to X-ray; (b) vitamins A and D, ultraviolet radiation, calcium by mouth and intravenously, and phosphorus by mouth; (c)

orthopedic measures designed to relieve local pressure and correct deformity.

The discussion includes a brief presentation of the relation of the parathyroids to osteomalacia and similar bone conditions.

The case reported was not due, apparently, to any abnormality of the parathyroids.

REFERENCES.

1. McCRUDDEN, F. H.: Arch. Int. Med., 1912, 9, 273.
2. A complete review of the literature as well as a report of a typical case is to be found in the article by I. SNAPPER of Amsterdam, Holland, published in the Arch. Int. Med., September, 1930, p. 506.
3. BARR, BULGER AND DIXON: Hyperparathyroidism. Jour. A. M. A., 1929, 92, 951.
4. Klinische Wochenschrift, 1930, 9, 1705.

DISCUSSION.

DR. GARVIN: I would like to ask if there is any medical way of controlling hyperparathyroidism? Is there any way of avoiding surgery?

DR. SNOWDEN: None that I know of; I do not know whether there is any way or not.